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Asymptomatic carotid bruit in patients who undergo coronary artery surgery

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Abstract. The clinical significance of the presence of carotid bruit was evaluated in 643 patients who underwent coronary artery surgery alone or in combination with other cardiac procedures. Carotid bruit was heard in 31 patients (5%) who were neurologically asymptomatic. All of them underwent coronary artery surgery without additional carotid procedures, and none of them developed neurological deficits during the postoperative period. Of the remaining 612 patients, 18 were identified as having a history of focal neurological disorders, and 9 of them had carotid bruit. All were analyzed by means of noninvasive tests and angiography. Five underwent carotid endarterectomy prior to (2 patients) and simultaneously with (3 patients) a coronary bypass procedure. Seven patients developed neurological deficits postoperatively. Most of the deficits were not lateralized or focal but diffuse, which suggests global cerebral ischemia not related to carotid disease. Only 1 patient had proven carotid obstructive disease and underwent successful carotid thrombendarterectomy 10 days postoperatively. This study, although based on limited material, supports the hypothesis that patients with asymptomatic carotid bruit can safely undergo coronary artery surgery. In the group of patients without neurological symptomatology, postoperative neurological deficits were rarely caused by occlusive carotid disease. However, patients with asymptomatic carotid bruit should be closely followed with the goal of identifying those who are at risk of developing neurological deficits. [Eur J Cardio-thorac Surg (1987) 1:11–15]

Key words: Carotid bruit – Coronary surgery – Neurological complications

Cerebrovascular accident is one of the most serious complications following coronary artery surgery (CAS). In spite of a continued decline in operative mortality for CAS, some recent studies reported an increase in the incidence of postoperative stroke in a certain subgroup of patients [6]. Still little is known about the etiology of neurological deficits after CAS. In the past, considerable attention has been paid to the relationship between the presence of cerebrovascular diseases and the occurrence of a stroke. Based on this correlation, different approaches have been advocated for the preoperative screening of patients who are candidates for CAS with regard to the presence of cerebrovascular disease and the treatment in cases of combined coronary and carotid pathology [5, 8–11, 13].

In 1983, after the evaluation of our own material, we introduced a standard protocol in our institute, consisting of a conservative approach to all neurologically asymptomatic patients (candidates for CAS), even in the presence of audible carotid bruit. This study is an evaluation of the patients in the subsequent 2 years.

Patients and methods

The hospital records of 643 patients who underwent coronary artery surgery alone or in combination with other cardiac procedures at the University hospital in Nijmegen in 1983 and 1984 were reviewed. During this period, a stable technique of extracorporeal circulation was applied, consisting of moderate hemodilution, general mild hypothermia, and left heart venting. Myocardial protection was achieved by using a crystalloid cardioplegic solution combined with local ice hypothermia. Patients with a carotid bruit as in high grade stenosis were tested by means of noninvasive techniques, regardless of the standard protocol consisting of a conservative approach to

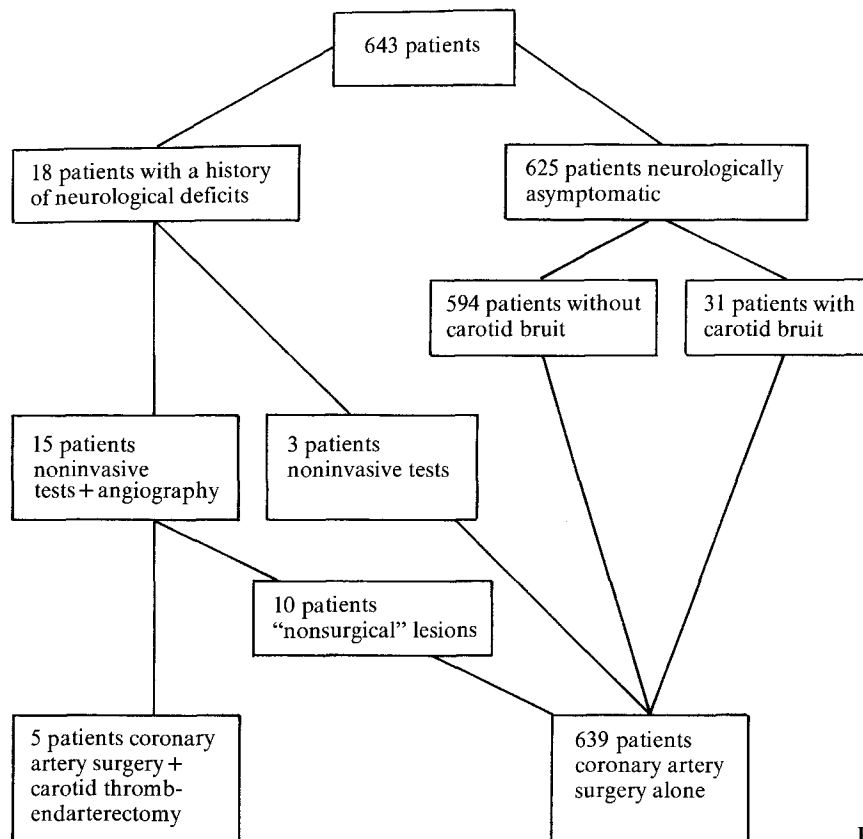


Fig. 1. Coronary artery surgery (1983–1984)

neurologically asymptomatic patients. The results of this test had no influence on the mode of treatment.

Carotid bruit was heard in 31 of the 643 patients (5%) who were neurologically asymptomatic (Fig. 1). All of them underwent coronary artery surgery without additional carotid procedures. Eighteen of the remaining patients were identified as having a history of neurological focal disorders (symptomatic patients). In this group, only 9 patients (50%) had carotid bruit. All 18 patients underwent preoperative assessment for cerebrovascular disease by means of oculopneumoplethysmography (Gee) and Doppler ultrasonography. Three patients from this group underwent only noninvasive evaluation for cerebrovascular disease. Two had a history of neurological signs 7 and 9 years earlier, followed by totally asymptomatic subsequent years. Noninvasive tests did not show any cerebrovascular obstructive disease. One patient had a history of neurological deficit but had negative noninvasive tests. Angiography was not performed because of the urgent necessity for coronary surgery (symptoms of impending infarction with severe three-vessel coronary artery disease). In the remaining 15 patients, the angiographic evaluation was performed as well as noninvasive tests. In 9 patients, angiography did not reveal lesions suitable for surgical treatment (lack of obstructive or ulcerative processes at the level of the carotid bifurcation).

In 1 patient, the internal carotid artery was totally occluded. Five patients (4 with high grade stenoses at the origin of the internal carotid artery and 1 with severe arteriosclerotic ulceration at the carotid bifurcation) underwent carotid endarterectomy (2 patients) prior to and (3 patients)

simultaneously with the coronary bypass procedure. In patients with stable angina, a staged procedure was performed. In patients with unstable angina or impending coronary occlusion, simultaneous procedures were carried out.

Results

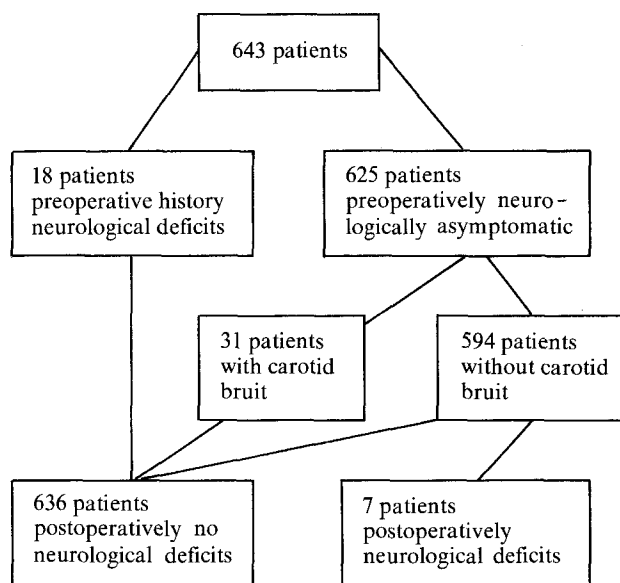
Eighteen patients had a history of neurological focal disorders (symptomatic patients) (Fig. 2). There was no mortality in this group, and none of the patients developed a neurological deficit in the postoperative period.

Six hundred twenty-five patients had no history of neurological focal disorders (asymptomatic patients). None of the 31 asymptomatic patients with audible carotid bruit developed neurological deficits after the coronary bypass surgery.

Of the remaining 594 patients, 7 developed neurological deficits perioperatively (Table 1). Two patients died: 1 after coronary artery surgery which was complicated by cerebral hemorrhage due to postoperative thrombocytopenia, the 2nd

Table 1. Postoperative neurological deficits after coronary artery surgery (7/643 patients)

Age (years)	Sex		
66	F	Thrombocytopenia; cerebral bleeding	Death
69	M	Renal insufficiency; peritoneal dialysis; peritonitis; mesenterial thrombosis – coma	Death
61	F	Vertebrobasilar insufficiency	Reversible
61	M	Global neurological disorders	Reversible
71	M	Left side hemiparesis; pulmonary insufficiency	Reversible
59	M	Left arm paresis	Reversible
64	M	TIA (10 days postop) (with anticoagulant therapy!) Angiography – 90% stenosis of the carotid artery	Carotid surgery – good

**Fig. 2.** Postoperative neurological deficits after coronary artery surgery

after coronary artery surgery and mitral valve replacement, which was complicated by renal insufficiency and mesenterial thrombosis. The patient became comatose and died 3 weeks postoperatively. Four patients developed transient neurological disorders, but extensive examination did not reveal any cerebrovascular disease. Only 2 patients had focal neurological deficits. One patient developed a transient ischemic attack 10 days after the coronary bypass procedure (while being treated with anticoagulants). Carotid angiography showed a high-grade stenosis of the ipsilateral internal carotid artery. The patient underwent carotid endarterectomy without subsequent complications. The remaining 587 pa-

tients did not develop any neurological deficits after coronary artery surgery.

Discussion

Many studies have been devoted to the preoperative identification of patients likely to develop neurological deficits after coronary artery surgery and the establishment of criteria for safe operative procedures especially with regard to extracorporeal circulation. Most attention has been concentrated on the presence of cerebrovascular disease in the screening procedure. The relationship between the presence of carotid lesions and strokes is rather well established, but little has been proven about this correlation as a complicating factor for coronary artery surgery.

In the screening of candidates for open-heart surgery, most attention has been paid to carotid bruit, which is considered an important diagnostic sign. Some authors consider the presence of carotid bruit to be an indication for arteriographic evaluation. On confirmation of the carotid lesion, an operation is advocated [4]. This approach is based on the supposition that, during coronary artery surgery, a critical reduction of the cerebral perfusion may develop distally to the carotid lesion. The loss of the characteristic pulsatile flow may also be an additional risk factor.

The incidence of carotid bruit in study populations (candidates for CAS) varies in different studies from 1.3% to 15% [1–3, 15, 19]. In our group of 643 patients, audible carotid bruit was present in 31 patients (5%). The clinical value of carotid bruit is highly controversial. In patients with high-grade stenoses, carotid bruit often cannot be heard. Therefore, lack of carotid bruit does not exclude the presence of carotid disease [2].

In some of the cases, severe obstruction or occlusion exists on the contralateral side, which is probably responsible for an increased bloodflow through the ipsilateral carotid artery [14]. Under optimal circumstances and with proper differentiation, carotid bruit is heard in 70%–90% of ipsilateral or contralateral carotid lesions in adults who are suspected of having cerebrovascular disease [14]. The ease with which carotid bruit can be detected and the extremely noninvasive character of this test are probably the reasons why so much attention and value are attributed to this sign.

In the history of coexisting carotid and coronary disease, a great deal of attention has been paid to the use of noninvasive tests, which have been suggested by many authors as ideal screening methods for identifying patients suitable for coronary artery surgery [10–12, 18]. These studies did not demonstrate a correlation between the presence of carotid disease documented by ultrasonic carotid duplex scan and the neurological outcome after coronary artery surgery. Some institutes continue to use arteriography for preoperative screening regardless of the considerable complications inherent to this method.

In the treatment of patients with identified carotid pathology who are candidates for coronary artery surgery, two approaches can be distinguished:

1. a one-stage procedure, in which carotid endarterectomy is performed simultaneously with CAS;
2. a staged procedure, in which carotid reconstruction is generally performed first.

Several studies in the late seventies suggested that the simultaneous procedure gives the best early and long-term results [4, 15]. Later publications from the Cleveland Clinic on large study groups have reported a higher incidence of stroke and mortality in patients who underwent simultaneous operations compared with staged carotid endarterectomy and CAS [8, 9, 11].

We consider patients with recent neurological symptoms as being at risk when undergoing CAS. Regardless of the presence of audible carotid bruit, they were subjected to noninvasive tests and arteriography. If the lesions detected were suitable for surgery, we considered the feasibility of carotid endarterectomy. In patients with stable angina, we prefer a staged approach, performing carotid reconstruction first. In patients with unstable angina or severe coronary pathology (i.e.,

lesions of the main coronary artery), we feel that the underlying cardiac problems justify simultaneous operations. We have not lost any patient in our study group, who was treated with the combined operative strategy.

The nature of postoperative neurological deficits as well as the circumstances in which they occur suggest certain conclusions. In our study of 1498 open-heart operations in adults (published in 1983) [18], we analyzed 19 patients who developed postoperative neurological symptoms. In the majority of cases, the neurological deficits were not focal but diffuse. In 12 of the 19 cases, neurological deficits occurred during or after an operation for valve replacement (mostly calcified aortic valve). It is also interesting that none of these 19 patients had an audible carotid bruit preoperatively. Moreover, most neurological complications occurred during the first 7 days postoperatively, not during open heart surgery itself. This may suggest that stroke is not caused solely by factors occurring during surgery but also by factors during the postoperative period, such as hypotension, hypoxia, or arrhythmia. In the series presented in this publication, only three of the seven patients presented focal neurological deficits and only one could be related to the presence of ipsilateral carotid obstructive disease.

These results coincide with several recent publications [6, 7, 10, 16] in that most of the neurological deficits occurring after CAS are not lateralized or focal but diffuse, suggesting global cerebral ischemia. Moreover, the majority of the patients with postoperative strokes did not have carotid bruit, a history of neurological disorders, or proven carotid obstructive disease. These data suggest that postoperative neurological deficits following CAS are rarely caused by occlusive carotid disease, but instead by emboli originating from the heart or aorta [17]. To prove this theory to an appropriate level of statistical significance (with a 95% degree of confidence), a study on a very large series of patients (approximately 10,000) would be necessary. At present, we do not have sufficient data to explain the exact cause of postoperative stroke [1, 6, 7, 16]. Gardner et al. [6] observed a decreasing operative risk after CAS (3.9%–2.6%) but an increasing risk of stroke (0.57–2.4%) over the years. The increased rate of strokes paralleled the increase in mean age of patients undergoing CAS, with an older age being the most significant risk factor of stroke. Less significant but also important were cerebrovascular disease, atherosclerosis of the ascending aorta, cardiopulmonary bypass time, and perioperative

hypotension. Heikkinen [7] suggested that previous neurological events also have an influence on the postoperative neurological status.

Based on our own observations with a limited number of patients and on data provided by others, we believe that an asymptomatic carotid bruit can be ignored in patients who are candidates for coronary artery surgery. However, Barnes et al. [3] have recently reported the high risk of transient ischemic attacks at long-term follow-up of patients with asymptomatic carotid disease. Therefore, such patients should be screened by means of noninvasive tests and, in the presence of the carotid disease, followed as closely as patients without coronary artery disease. At present, we feel that patients with a recent history of neurological focal disorders should be screened preoperatively for the presence of occlusive carotid disease. The patients with proven significant carotid disease should be treated with a staged or combined procedure, depending on the severity of the underlying cardiac pathology.

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